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l6 and (cutaneous t cell lymphoma or ctl)	14

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DB Name	Query	Hit Count	Set Name
USPT	l6 and (cutaneous t cell lymphoma or ctl)	14	L7
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USPT	l3 and (augment? or increase? or enhance?)	253	L4
USPT	l1 and l2	266	L3
USPT	interferon gamma	1841	L2
USPT	interleukin 12 or IL-12	581	L1

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FILE 'MEDLINE, BIOSIS, CANCERLIT, CAPLUS, EMBASE' ENTERED AT 13:50:29 ON 29 SEP 2000

L1	22703	S	INTERLEUKIN 12 OR IL 12
L2	141368	S	INTERFERON GAMMA OR IFN-GAMMA
L3	55367	S	L2 AND (PRODUCTION OR STIMUALTION OR FORMATION)
L4	6848	S	L1 AND L3
L5	3222	S	L4 AND HUMAN
L6	831	S	L5 AND (TREAT? OR ADMINISTER? OR INJECT?)
L7	41	S	L6 AND (CUTANEOUS T CELL LYMPHOMA OR CTL)

ANSWER 14 OF 14 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 95058274 EMBASE
DN 1995058274
TI The application of **IL-12** to cytokine therapy for tumors.
AU Nishimura T.
CS Department of Immunology, Tokai University School of Medicine, Boseidai,Isehara 259-11, Japan
SO Biotherapy, (1995) 9/1 (16-26).
ISSN: 0914-2223 CODEN: BITPE
CY Japan
DT Journal; Article
FS 016 Cancer
026 Immunology, Serology and Transplantation
037 Drug Literature Index
LA Japanese
SL Japanese; English

=> d 14 ab

L8 ANSWER 14 OF 14 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AB **IL-12**, which is produced by antigen presenting cells such as M.diameter. and B cells, has been considered a novel cytokine useful for cytokine tumor therapy. In this paper, we investigated the potentiating effect of **IL-12** on the induction of antitumor effector cells in both **human** and mouse system. Culture of **human** CD4+ T cells with immobilized anti-CD3 mAb in the presence of IL-2 and **IL-12** caused their skewing to Th1 type of Th cells. In addition, we demonstrated that **IL-12** could markedly stimulate the generation of **IFN-.gamma.** high producing CD8+ killer T cells from **human** PBMC. In general, the generation of **CTL** reactive against autologous tumor cells is very difficult. However, **IL-12** was demonstrated to enhance the generation of tumor-infiltrating lymphocyte (TIL)-derived CD8+ **CTL** reactive against autologous tumor cells. **Treatment** of mice with ip **injection** of small dose of **IL-12** caused in vivo **production** of **IFN-.gamma.** and the generation of LAK cells. In parallel with these, **IL-12** showed a potent antitumor activity against ip **injected** MBL-2 T lymphoma cells, but not IL-2. Moreover, it was demonstrated that **IL-12** inhibited the growth of id **injected** tumor by systemic administration. Using primary tumor induction system, we initially found that **IL-12** could inhibit the incidence of chemically-induced papilloma **formation** in c-Ha-ras-transgenic mice. We also discussed the usefulness of **IL-12** in gene therapy of tumor from the evidence that **IL-12** gene transfer into tumor cells completely abrogates their tumorigenicity.

=> d 6 bib ab

L8 ANSWER 6 OF 14 MEDLINE DUPLICATE 5
AN 1999265561 MEDLINE
DN 99265561
TI Retinoids synergize with interleukin-2 to augment **IFN-**

gamma and **interleukin-12 production**
by **human** peripheral blood mononuclear cells.

AU Fox F E; Kubin M; Cassin M; Niu Z; Trinchieri G; Cooper K D; Rook A H
CS Department of Dermatology, University of Pennsylvania School of Medicine, Philadelphia 19104, USA.

NC CA 58841 (NCI)
CA 20833 (NCI)
CA 32898 (NCI)
+
SO JOURNAL OF INTERFERON AND CYTOKINE RESEARCH, (1999 Apr) 19 (4) 407-15.
Journal code: CD4. ISSN: 1079-9907.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199909
EW 19990903

AB We have demonstrated previously that cells from both the skin and peripheral blood from patients with **cutaneous T cell lymphoma** (CTCL) have elevated levels of protein and mRNA for Th2 cytokines, interleukin-4 (IL-4) and IL-5, and depressed levels of Thl cytokines, IL-2 and **interferon-gamma** (**IFN-gamma**). Furthermore, **IL-12** in vitro can restore **IFN-gamma production** by these patients' cells to near normal levels. Because retinoids exert therapeutic activity in CTCL and are potent modulators of growth and differentiation of hematopoietic cells, we investigated the role of retinoids in modulating Thl cytokine **production**. Peripheral blood mononuclear cells (PBMC) from normal donors and patients with CTCL were cultured with medium, IL-2, 13-cis-retinoic acid, all-trans-retinoic acid, acetretin or etretinate alone, or IL-2 plus the retinoids for 24 h, and levels of **IFN-gamma** were determined using ELISA. IL-2 or retinoids alone could induce low but significant levels of **IFN-gamma**. However, when IL-2 was cultured with each retinoid, a synergistic augmentation of **IFN-gamma** levels (4-fold to 90-fold) was observed except in the case of etretinate. All-trans-retinoic acid (ATRA) was the most potent IFN-y inducer. Similar studies performed using PBMC from CTCL patients indicated the **IFN -gamma** augmentation occurred but in a blunted manner. The IFN-y-inducing effect of ATRA and 13-cis-retinoic acid could be abrogated by addition of anti-**IL-12** antibodies, suggesting that **IL-12** plays a role in the synergistic upregulation of **IFN-gamma**. Using an **IL-12** p40-specific radioimmunoassay (RIA), we confirmed the presence of **IL-12** in IL-2 plus retinoid-**treated** culture supernatants. Purified monocytes cultured with IL-2 plus ATRA did not secrete **IL-12**. Only when monocytes were cocultured with lymphocytes was there an increase in **IL-12** **production**, suggesting the involvement of a paracrine feedback loop requiring both monocytes and lymphocytes. These data suggest that retinoids can induce Th1 cytokines from normal and CTCL PBMC and that this induction may be mediated through **IL-12**